Review

Mesenteric Ischaemia

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Introduction

Mesenteric ischaemia occurs infrequently and can be life threatening. The mortality rate is high in patients with acute arterial occlusion.[1] Most cases of acute mesenteric ischaemia involve either acute arterial thrombosis or embolization of the superior mesenteric artery.[2] Emboli are usually found 3 to 10 cm distal to the origin and just past the origin of the middle colic artery.[3] Other causes of acute ischaemia are less common.[4,5] Symptoms of acute intestinal ischaemia and possible infarction of the intestines are caused by the sudden reduction in blood flow to the bowel. The mortality rate of an acute occlusion is high, and ranges from 60% to 100%.[5] The risk of atherosclerotic-related mesenteric occlusive disease has become more prevalent in the population because life expectancy has increased. Aged patients with severe cardiovascular disease are at the highest risk. Females also have a slightly higher risk than males.

A rapid onset of symptoms usually occurs after severe embolization of the superior mesenteric artery, and patients seek help promptly. Patients have sudden abdominal pain and the severity of pain is usually much out of proportion to the physical findings.[3] Most patients have been vomiting and/or defaecating, and have leukocytosis. Patients have tenderness in the right lower quadrant and hyperactive bowel sounds. Later developments are distension of the abdomen, progressive necrosis from the intestinal villus tips to the peritoneum, oliguria, metabolic acidosis, elevated hepatic enzymes, hyperamylasaemia and shock. Although magnetic resonance (MR) angiography and computed tomography (CT) can image the mesenteric vessels and locate the source of the disease, any delay in revascularization can lead to grave consequences.[6] Mortality is high even with treatment. The appropriate treatment usually can be determined from the appearance of the bowel and the absence of a pulse in the arteries.[2] In the early stage of acute obstruction, the bowel is not necrotic but has a dull gray appearance, without the normal sheen. If the vasa recta are pulseless, it is important to determine the viability of the bowel. When the superior mesenteric artery is thrombosed, the pulse is frequently absent. An embolus at, or distal to, the origin of the middle colic artery usually spares the proximal jejunum and an embolectomy usually will restore the circulation. Nonviable bowel must be resected and the revascularization procedure should achieve pulsatile blood flow in the mesenteric arteries. It is clear that the signs of bowel infarction, such as peritonitis, deterioration of haemodynamics, and metabolic acidosis indicate the need for an emergency laparotomy.[7]

When symptoms develop gradually, patients usually wait longer before seeking help, and they have a higher mortality rate as a result.[7] Commonly, patients with thrombosis have a slow progression of symptoms, which is related to the degree of bowel perfusion. Blood flow in the intestine can vary from 20% when fasting to 35% after eating. The bowel rarely becomes ischaemic when only one artery is involved, because collateral circulation exists at multiple levels and adequate intestinal perfusion can be maintained at near normotensive blood pressures. Symptoms are present when there are multiple occlusions that cause a significant fall in blood pressure. The bowel becomes ischaemic when the blood pressure is less than 40 mmHg, and the extent of damage is determined by the duration of hypoxia.[1] Without revascularization and resection, bowel necrosis, perforation, sepsis, multiorgan failure and death ensue.

Patients who are haemodynamically stable, without evidence of peritonitis and significant abdominal pain, should have a diagnostic study to demonstrate occlusion of the mesenteric vessels. A colour duplex scan of the mesenteric vessels can rule out occlusive disease. MR angiography is useful to determine the location and extent of occlusions

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before planning revascularization. Prior to the revascularization, haemoconcentration and blood pressure should be corrected, if possible, by replacing fluids, an effort made to correct acidosis and heparin given. Tissue injury can occur within 3 hours of the bowel becoming ischaemic, and can lead to multiple organ dysfunction and death. Acute intestinal ischaemia requires surgical intervention without delay. While an expeditious embolectomy can readily restore the circulation, it is often difficult to achieve an adequate thrombectomy of the splanchnic arteries. Infarctions usually extend further with acute arterial thrombosis than with an embolus, and can extend from the duodenum to the transverse colon. Many vascular surgeons prefer to perform antegrade or retrograde bypasses rather than a thrombectomy. When the graft can be made to the superior mesenteric artery to restore blood flow to the ischaemic bowel, revascularization of other splanchnic vessels is probably not required.[8] Percutaneous transluminal angioplasty of the superior mesenteric artery, celiac trunk or inferior mesenteric artery can also restore the circulation.[9,10] Intra-arterial fibrinolysis with urokinase or tissue plasminogen activator may be the best option for some high-risk patients.[11] Since vasoconstriction and hypoxia can complicate recovery after revascularization, vasodilators may be beneficial to improve blood flow.[5] After ischaemic damage, the restoration of oxygenated blood to the hypoxic bowel may cause further injury from oxygen-free radicals. The administration of superoxide-free radical scavengers may prevent reperfusion injury.[12]

Non-occlusive mesenteric ischaemia

Non-occlusive mesenteric ischaemia occurs from severe microvascular vasoconstriction, and usually no vascular occlusion can be demonstrated because pulsatile blood flow is present in larger arteries.[13] Unfortunately, symptoms are vague and the diagnosis is difficult. It has been suggested that non-occlusive mesenteric ischaemia may be the underlying cause in 20% to 30% of patients with acute mesenteric ischaemia and is the cause for 0.1% of all hospital admissions.[14] The aetiology is multifactorial, including persistent vasoconstriction, vasospasm, intestinal hypoxia from low cardiac output, ischaemia-reperfusion injury, increased intestinal metabolic demand and infection.

Patients with severe cardiac failure can also develop non-occlusive mesenteric ischaemia from the vasospasm due to an elevated sympathetic activity or from hypovolaemia related to maintaining adequate cardiac and cerebral perfusion. The autoregulation of the splanchnic blood flow can be compromised by sympathetic nerve stimulation, circulating catecholamines and medications such as digitalis. Patients may have abdominal distension, abdominal tenderness, hypotension, leukocytosis, fever, diminished bowel sounds, nausea and vomiting, and metabolic acidosis. Bowel ischaemia can be reversed by papaverine infusion and the resection of necrotic bowel. A risk of vasospasm can occur in elderly patients with acute myocardial infarction, congestive heart failure, dysrhythmia, sepsis or hypovolaemia. Delays in the diagnosis are a frequent cause of the rapid deterioration of these patients, and many expire before surgery can be performed. Both early diagnosis and prompt surgical intervention are necessary for survival of these patients.

Chronic intestinal ischaemia

Chronic intestinal ischaemia is caused by the gradual reduction in blood flow to the intestine and typically causes postprandial abdominal pain and weight loss. It occurs more frequently in older women.[15] The most common cause of chronic intestinal ischaemia is atherosclerotic-related stenosis of the mesenteric arteries. A stenosis of greater than 50% is present in 18% of patients older than 65 years,[16,17] but very few patients have symptoms. Most patients develop sufficient collateral circulation to the intestine to prevent ischaemic symptoms. When the superior mesenteric artery is occluded, the pancreaticoduodenal arteries supply blood via the hepatic and gastroduodenal arteries to the bowel. When the coeliac artery is occluded, the inferior mesenteric artery supplies blood to the small bowel via the left colic branch. A large meandering mesenteric artery, an important vessel in the collateral circulation from the inferior mesenteric artery, is frequently seen. Symptoms usually occur if two or more vessels are occluded. The peak systolic blood flow velocities determined by colour duplex ultrasonography at several sites along the course of the vessels can be diagnostic. While the superior mesenteric artery usually can be imaged in most patients, the coeliac artery may not be adequately visualized in 20% of patients.[18]

Quantifying the blood flow of the inferior mesenteric artery is usually difficult. Contrast-enhanced MR angiography has now become an essential technique for diagnosing an occlusion and determining the collateral circulation of the coeliac and superior mesenteric arteries.[6] Single-vessel bypasses to the superior mesenteric artery have been very successful in these patients, who usually have multiple-vessel occlusions. In

one study, the procedure had a perioperative mortality rate of 3% for chronic ischaemia, a 5-year survival rate of 61% and a 9-year assisted primary graft patency of 79%.[19] Bypass grafts have been antegrade from the abdominal aorta, retrograde from the aorta, the common iliac artery or previous grafts. Those who advocate multiple-vessel revascularization indicate that there is a higher incidence of graft failure and recurrence of symptoms.[20,21] Palmaz stents have been used in the proximal coeliac artery to completely resolve abdominal angina.[22,23] When the results of open surgery were compared to percutaneous angioplasty and stent placement, percutaneous angioplasty had a higher incidence of recurrent symptoms.[24] Antegrade aortocoeliac bypass and transaortic endarterectomy have been used for older or poor-risk patients, and are usually adequate for multiple outflows. Antegrade mesenteric bypass grafts from the distal thoracic aorta have also been associated with low mortality and morbidity rates.[25] Although antegrade bypasses to the supracoeliac aorta as the inflow source are usually preferred to establish adequate inflow and reduce possible kinking, compression, turbulence or thrombosis, they have been associated with renal ischaemia and can be technically challenging. Retrograde bypasses usually arise from the infrarenal artery or iliac artery, and have the advantage of technical ease and avoiding renal ischaemia, but have been related to lower inflow and kinking. Revascularization of the superior mesenteric artery and coeliac axis may minimize the recurrence of symptoms and organ infarction if one graft fails.[26]

Graft failures may be higher in men.[27] A postoperative assessment of the revascularization is necessary in patients who still may have nonviable bowel. After 24 to 48 hours, a "second-look" laparotomy can confirm whether the bowel is healthy.[28] While some surgeons advocate that all patients should have a second look, others selectively use an exploratory laparotomy only for patients who do not improve. In either case, it is important to evaluate the progress of the patient for at least 48 hours postoperatively.

Mesenteric venous thrombosis

Mesenteric venous thrombosis can occur either spontaneously as a primary event, or secondarily as a consequence of cancer, hypercoagulation, cirrhosis, splenomegaly, infection, trauma, pancreatitis or diverticular disease.[3,29,30] It can be asymptomatic or cause an infarction of the bowel. The disease has been classified according to the duration of symptoms. The acute form typically is associated with progressively severe abdominal pain, and nausea and vomiting of less than 4 weeks' duration. The chronic form presents with vague abdominal pain or distension of greater than 4 weeks' duration and without bowel infarction, or is incidentally found on abdominal imaging. The acute form comprises nearly 5% to 15% of all acute mesenteric ischaemic cases.[29]

The initial diagnostic test for mesenteric venous thrombosis should be duplex ultrasonography of the mesenteric vessels. CT may be the definitive test because it has the highest sensitivity for a diagnosis. Conventional angiography and abdominal radiography provide little value for a reliable diagnosis. After embolectomy or thrombectomy and bowel resection, the risk of recurrent mesenteric ischaemia is low when appropriate anticoagulation therapy is provided. The mortality rate is approximately half of that of mesenteric arterial obstructive disease.[31]

Superior mesenteric artery trauma

Trauma to the superior mesenteric artery also results in a high mortality from the complications of intestinal infarction or ischaemia, failure of the repair, short bowel syndrome or graft thrombosis. Mortality is associated with the severity of the ischaemia-grade of Fullen's anatomic classification of superior mesenteric artery injury, the American Association for the Surgery of Trauma-Organ Injury Score (AAST-OIS), high intraoperative transfusions, acidosis and dysrhythmias.[32] Usually, a ligation is done in these patients. Superior mesenteric artery injuries are highly lethal. The outcome is similar to penetrating and blunt trauma.

Summary

Survival after mesenteric ischaemia depends upon the expeditious restoration of the circulation and the resection of nonviable bowel. Of the utmost importance are an early diagnosis and the timely reperfusion of the bowel. Therefore, the initial clinical evaluation is very critical, and it is vital to ascertain if time is available to obtain diagnostic tests such as MR angiography or CT to help plan the revascularization. In spite of emergency diagnostic and therapeutic techniques performed by vascular surgeons, the mortality rate remains high for acute intestinal ischaemia.

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